---31. (new) A method for reducing ß-cell dysfunction in an individual with a pancreatic disorder, wherein said dysfunction results in diabetes, comprising:

(i) introducing a nucleic acid molecule encoding an inhibitor of IL-1 β into a β cell; and

(ii) transplanting the β cell of step (a) into the individual so as to reduce β cell dysfunction.

32. (new) The method of claim 31 wherein the inhibitor of IL-1 β activity is an interleukin-1 receptor antagonist protein.

33. (new) The method of claim 31 wherein the inhibitor of IL-1 β activity is an NF-kß inhibitor.

34. (new) The method of claim 31 wherein the inhibitor of IL-1 β is an insulin like growth factor-1.

35. (new) A method for reducing Fas mediated ß-cell apoptosis in an individual with a pancreatic disorder, wherein said β-cell apoptosis results in diabetes, comprising:

(i) introducing a nucleic acid molecule encoding an inhibitor of Fas mediated apoptosis into a β cell; and

(ii) transplanting the β cell of step (a) into the individual so as to reduce β cell apoptosis.

36. (new) The method of claim 35 wherein the inhibitor of Fas mediated apoptosis is an dominant negative mutant of the Fas protein.

37. (new) The method of claim 35 wherein the inhibitor of Fas mediated apoptosis is a dominant negative mutant of the FADD protein.

38. (new) The method of claim 35 wherein the inhibitor of Fas mediated apoptosis is a member of the bcl-2 protein family.

39. (new) A mammalian ß-cell comprising a recombinant nucleic acid molecule, said nucleic acid molecule comprising and expressing an inhibitor of IL-1 β activity, wherein the expression of the inhibitor of IL-1 β activity reduces said β cell dysfunction.

40. (new) The ß-cell of claim 39 wherein the inhibitor of IL-1 β activity is an interleukin-1 receptor antagonist protein.